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Role of intra-aortic balloon pump counterpulsation in the treatment of acute myocardial infarction complicated by cardiogenic shock: Evidence from the Portuguese nationwide registry

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Abstract

Background: In previous guidelines, intra-aortic balloon pump (IABP) use was strongly recommended in the treatment of cardiogenic shock in the context of acute myocardial infarction. The recent IABP-SHOCK II trial demonstrated no benefit in short- and medium-term mortality with the use of IABP. It was our objective to evaluate in a real life nationwide population of patients with acute myocardial infarction the impact of IABP in short- and medium-term mortality.

Methods: We included patients admitted with acute myocardial infarction in Killip class IV in the first 24 hours, all submitted to urgent coronary angiography. Our study objective was the occurrence of hospital and six-month all-cause mortality.

Results: From the 33,300 patients included in the registry, 4.2% presented with Killip class IV in the first 24 hours and 646 (43.6%) were submitted to urgent coronary angiography. IABP was implanted in 19.8% of these patients. The IABP group was younger, had higher admission heart rate, more multivessel disease and more left main disease. There were 260 hospital deaths (40.2%), similar between groups (46.1% *vs.* 38.8%, p=0.132). IABP use was associated with a deleterious effect in patients with previous MI and beneficial effect in patients with mechanical complications. IABP use had a neutral effect on mortality (hazard ratio 1.14, 95% confidence interval 0.84–1.56). This was further confirmed in a propensity score matching analysis.

Conclusions: In a real life population of patients with acute myocardial infarction, the use of IABP for the treatment of cardiogenic shock was associated with a neutral effect.

Keywords

Intra-aortic balloon pump, cardiogenic shock, acute myocardial infarction

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Introduction

Cardiogenic shock is a rare complication, but a deadly one in patients with acute myocardial infarction. In previous registries, which represents real-world practice, the rate of cardiogenic shock in populations with acute coronary syndromes (ACS) ranged from 1% to 4.6%.[1,2] Despite limited evidence from randomized controlled trials, intra-aortic balloon pump (IABP) counterpulsation was recommended

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as class I treatment in patients with infarct-related cardiogenic shock in previous guidelines of the American Heart Association/American College of Cardiology and the European Society of Cardiology and is the most commonly used mechanical assistance device in that context.[3,4]

More recently, there was a suggestion from several meta-analysis that the use of IABP might not be associated with any survival improvement, although not all results were identical.[5,6] More recently, in the contemporary era of myocardial infarction treatment, the IABP-SHOCK II trial showed no benefit with the use of IABP in patients with cardiogenic shock.[7] However, these results do not always reflect real-world treatment practice and sometimes the translation to real-life populations is not always adequate.

The aim of our study was to assess the role of IABP treatment in patients with acute myocardial infarction complicated by cardiogenic shock from a large nationwide registry of ACS. This registry reflects real-life treatment and we intended to study the potential beneficial or harmful effects of IABP.

Methods

We included consecutive adult patients (⩾ 18 years) from the Portuguese Registry on Acute Coronary Syndromes (ProACS) between 1 January 2002 and 31 October 2013. This registry is a continuous, prospective and observational registry, with 33 participating cardiology departments from Portugal (continental and islands).[8,9] Acute myocardial infarction (MI) diagnosis was defined according to the universal definition criteria for type 1 myocardial infarction.[10] Inclusion criteria in the registry were a history of chest pain at rest or other symptoms suggestive of an ACS (the most recent episode occurring within 48 h of admission) with or without new or presumed new significant ST-segment–T wave changes/new left bundle branch block or elevated levels of biomarkers of myocardial damage with a rise and/or fall of these markers. A persistent (> 30 min) ST-segment elevation was considered ST elevation MI (STEMI). All other cases with elevated levels of biomarkers of myocardial damage were considered non-STEMI (NSTEMI).

For the purpose of the present study, only patients admitted in Killip–Kimbal class IV or who developed it in the first 24 h after admission with early revascularization planned were selected. Cardiogenic shock was defined by a systolic blood pressure of less than 90 mmHg for more than 30 min or if the patient needed infusion of catecholamines to maintain a systolic blood pressure above 90 mmHg, had clinical signs of pulmonary congestion, and had impaired end-organ perfusion.

Data was registered in a dedicated computer database, including demographic, clinical and patient management-related characteristics, as well as clinical outcomes. Hypertension, diabetes and hyperlipidaemia were defined as either previously known or on specific therapy. If the patients had smoked during the previous 30 days they were classified as smokers and were self-reported.

Decisions on patient management strategy, including referral for coronary angiography and performance/mode of myocardial revascularization (either percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG)) were left to the attending physician and the site-specific protocols. Significant coronary artery stenosis was defined as more than 50% of luminal obstruction.

Our primary end point was all-cause mortality during the index hospitalization. We also considered other secondary end points: in-hospital re-infarction, mechanical complications, cardiac arrest and the composite end point of death/re-infarction. Recurrent chest pain for more than 20 min with new ECG changes and/or a new increase in biomarkers of myocardial necrosis (increase > 20% in troponin compared with previous levels) were considered a re-infarction. Six-month all-cause mortality was also assessed.

We also considered some safety end points: stroke/transient ischaemic attack (TIA) and major bleeding. Stroke/TIA was defined by the presence of new neurological symptoms with associated signs of ischaemia or bleeding on computed tomography or magnetic resonance imaging. Major bleeding was evaluated according to the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO) criteria – intracranial bleeding or significant bleeding with haemodynamic compromise requiring specific intervention.[11]

The study complies with the 1975 Declaration of Helsinki. The ethics committee for clinical research approved the study and patients gave their informed consent. This registry is registered at ClinicalTrials.gov with the identification number: NCT01642329.

Statistical analysis

Categorical variables are reported as percentages and continuous variables are reported as mean ± standard deviation. Differences between groups for discrete variables were tested with the chi-squared test or Fisher's exact test as appropriate. We used the two-tailed Student's *t*-test to compare continuous variables. Continuous variables without normal distribution are reported as median and inter-quartile range and were compared with the Mann–Whitney test.

Multivariable logistic regression models (with forward stepwise selection) were used to assess the association of IABP therapy with all-cause in-hospital mortality and the composite end point death/re-infarction. Variables were removed from the model when the *p*-value exceeded 0.10. Factors that remained significant at the 0.05 level in the multivariable models were considered to be significant contributors and were kept in the model. Potential confounding factors offered to the logistic regression models included: age, gender, diabetes, hypertension, smoking status, hyperlipidaemia, renal failure, prior MI, prior revascularization, prior stroke, STEMI diagnosis, multi-vessel

disease. The estimates of the association between predictors and end points are presented as odds ratios (ORs) with 95% confidence intervals (CIs).

For all-cause in-hospital mortality and the composite end point death/re-infarction, specific subgroup analysis was performed in subgroups defined according to gender, age (< 65 years, 65–74 years, or ⩾ 75 years), presence or absence of diabetes, STEMI versus NSTEMI, with or without mechanical complications, and previous or no previous MI. The interaction analysis between IABP therapy and each subgroup variable was performed considering multivariable logistic regression models.

We also performed a propensity score matching analysis to further adjust for the non-randomized assignment of patients to treatment and for the potential bias due to differences in both study groups. A propensity score was calculated for each individual by logistic regression as the likelihood of assignment to treatment with an IABP. The propensity model consists of the following variables: age; gender; hypertension; diabetes; hyperlipidaemia; smoking status; previous PCI, CABG, MI, stroke/transient ischaemic attack and peripheral arterial disease; ACS type; admission by the emergency medical system; admission heart rate and systolic blood pressure; in-hospital treatment with acetyl-salicylic acid, clopidogrel, angiotensin converting enzyme (ACE) inhibitor, beta-blocker and statins; left main disease; multivessel disease; and in-hospital PCI or CABG. Then we performed a 1 to 1 or 1 to 2 (whenever possible) matched analysis on the basis of the estimated propensity score of each patient. A standardized difference of less than 5% supports the assumption of balance between the two groups. After this propensity score matching, baseline and in-hospital characteristics were compared.

Estimates of event-free survival at six-month follow-up were calculated by the Kaplan–Meier method and curves were compared with the log-rank test. We used a Cox proportional-hazards regression model with the p level for entry into and removal from the model set at 0.05 and 0.10, respectively (with a forward stepwise with likelihood ratio statistic method) for selecting independent predictive variables for all-cause mortality. The estimates of the association between predictors and endpoints are presented as hazard ratios (HRs) with 95% CI. This analysis was applied in both the baseline study groups as well as in the propensity score matched groups.

For all statistical analysis, we used the IBM SPSS statistics software package (version 19.0.0.2). All statistical tests were two-sided with a critical value of 0.05 for statistical significance.

Results

From a total of 33,300 patients included in the registry during the study period, 1481 (4.2%) developed cardiogenic shock on admission or in the first 24 h after admission (Figure 1). From these, 646 patients (43.6%) underwent coronary angiography in the first 24 h and were selected for the present analysis. Excluded patients were older, less frequently males, with fewer risk factors and previous PCI (despite a higher rate of previous MI). They also presented less frequently with STEMI and had lower heart rate and a worse Global Registry of Acute Coronary Events (GRACE) risk score (Supplementary Material Table 1 online). They received less medication and less primary PCI, with higher rates of thrombolysis and mortality. In this population, 22.7% died very early (< 24 h after admission) (*vs*. 15.6% in the study population, $p<0.001$) and 51.0% were initially admitted in a hospital without catheterization facilities (*vs*. 29.7%, $p<0.001$), which might have been one possible explanation for not being submitted to early invasive strategy.

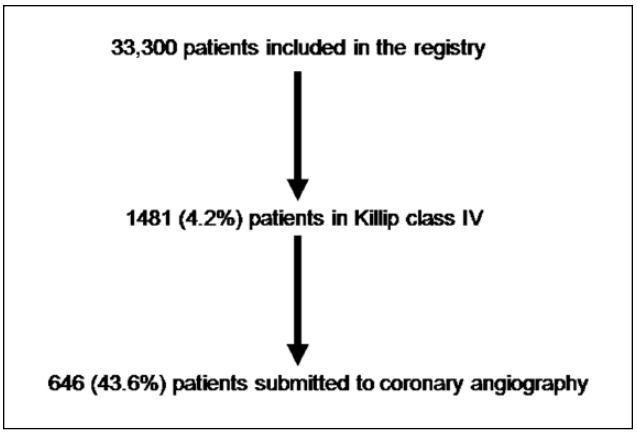

Figure 1. Flowchart of patient selection for inclusion in the study.

The mean age of our study population was 69 ± 12 years, 65% males and only 19.8% received IABP therapy. Patients in the IABP group were younger (Table 1). They were less often referred by the emergency medical system and had higher heart rate on admission. On coronary angiography they had more often multivessel or left main disease. The proportion of patients with STEMI was similar between both groups; however, anterior myocardial was more often present in patients that received IABP. All other baseline characteristics are well balanced between the two groups (Table 1).

The procedure most often used for revascularization was primary PCI in patients with STEMI and also PCI in NSTEMI patients. In the index hospitalization, only 2.4% of patients underwent CABG and in 16% of the patients no revascularization procedure was performed. Concomitant medications are shown in Table 1 and were also well balanced, with the exception of a trend to a lower use of acetyl salicylic acid and higher use of statin in the group without IABP.

Primary and secondary end points

In-hospital mortality rate was 40.2%, slightly higher in the group that received IABP (OR 1.35, 95% CI 0.91–1.99, p=0.132) (Table 2). Also re-infarction was slightly higher in the group with IABP (OR 2.11, 95% CI 0.96–4.63,

Table 1. Clinical characteristics by study group.

	Without IABP *n*=518	With IABP *n*=128	*p*-value
Age, years	69 ± 12	65 ± 12	0.001
Male gender, %	65.0	67.2	0.593
BMI, kg/m^2	26.6 (24.2–29.1)	26.3 (24.5–28.4)	0.761
Risk factors, %			
Hypertension	61.9	62.2	0.943
Hyperlipidaemia	42.9	46.8	0.431
Diabetes	29.6	33.1	0.453
Smoking	24.1	31.5	0.086
Previous history, %			
Myocardial infarction	14.8	10.2	0.177
PCI	10.1	7.8	0.437
CABG	2.5	0.8	0.323
Stroke/TIA	9.1	10.2	0.715
PAD	6.8	3.1	0.115
Initial presentation			
EMS, %	22.7	12.5	<0.001
Heart rate, beats/min	81 ± 28	91 ± 29	0.001
SBP, mmHg	108 ± 38	103 ± 31	0.145
Killip class IV, admission, %	47.8	51.2	0.199
STEMI, %	82.6	80.5	0.429
Anterior	51.9	71.8	<0.001
Thrombolysis	12.2	10.2	0.607
Primary PCI	87.8	89.8	0.618
Laboratory data[a]			
Creatinine, mg/dl	1.2 (0.9–1.6)	1.2 (1.1–1.6)	0.323
eGFR, ml/min per 1.73m^2	55 (37–76)	60 (41–82)	0.692
Blood glucose, mg/dl	174 (133–260)	219 (156–280)	0.075
GRACE risk score	190 (161–213)	192 (150–218)	0.751
Multivessel disease, %	56.0	63.0	0.014
Left main disease, %	11.6	25.1	<0.001
Treatment, %			
ASA	94.5	98.4	0.061
Clopidogrel	83.7	85.9	0.540
ACEI	53.1	49.6	0.486
Beta-blocker	41.8	41.3	0.912
Statin	79.3	72.2	0.087
PCI	82.5	87.3	0.190
CABG	2.0	4.0	0.092

[a]Data from only the 223 patients included since 1 October 2010 (second phase of the ProACS registry).
IABP: intra-aortic balloon pump; BMI: body mass index; PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting; TIA: transient ischaemic attack; PAD: peripheral arterial disease; SBP: systolic blood pressure; EMS: emergency medical system; STEMI: ST elevation myocardial infarction; eGFR: estimated glomerular filtration rate; GRACE: Global Registry of Acute Coronary Events; ASA: acetyl salicylic acid; ACEI: angiotensin converting enzyme inhibitor.

Table 2. Outcomes and complications in both study groups.

	Without IABP *n*=518	With IABP *n*=128	*p*-value
All-cause hospital mortality, %	38.8	46.1	0.132
Hospital re-infarction, %	3.9	7.8	0.057
Death/re-infarction	40.3	50.8	0.032
Mechanical ventilation, %	24.5	63.3	<0.001
Mechanical complication, %	9.3	9.4	0.893
Cardiac arrest, %	23.4	28.1	0.260
Stroke/TIA, %	2.1	2.3	0.746
Major bleeding, %	6.2	8.6	0.326

IABP: intra-aortic balloon pump; TIA: transient ischaemic attack.

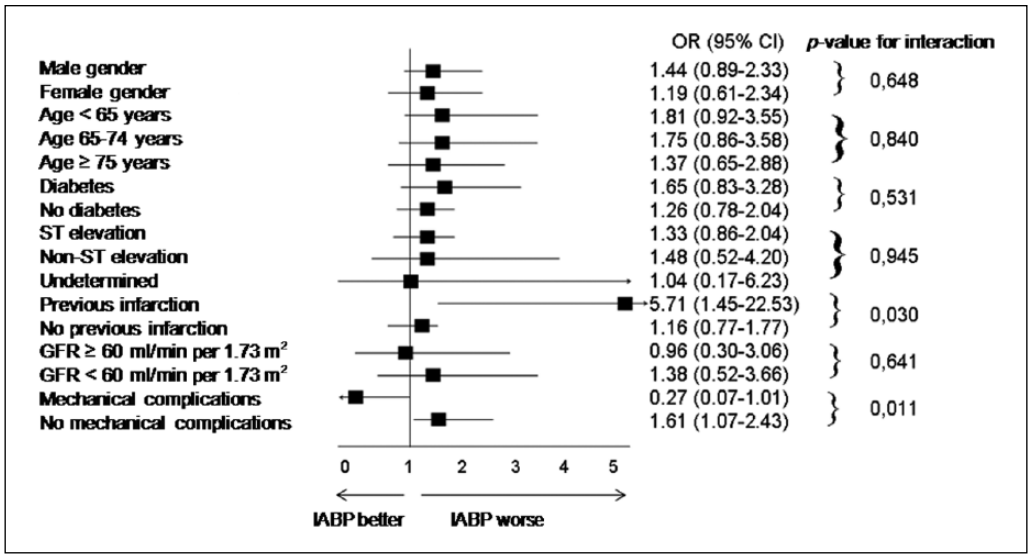

Figure 2. Subgroup analysis for the primary end point (all-cause in-hospital mortality).
OR: odds ratio; CI: confidence interval; GFR: glomerular filtration rate; IABP: intra-aortic balloon pump.

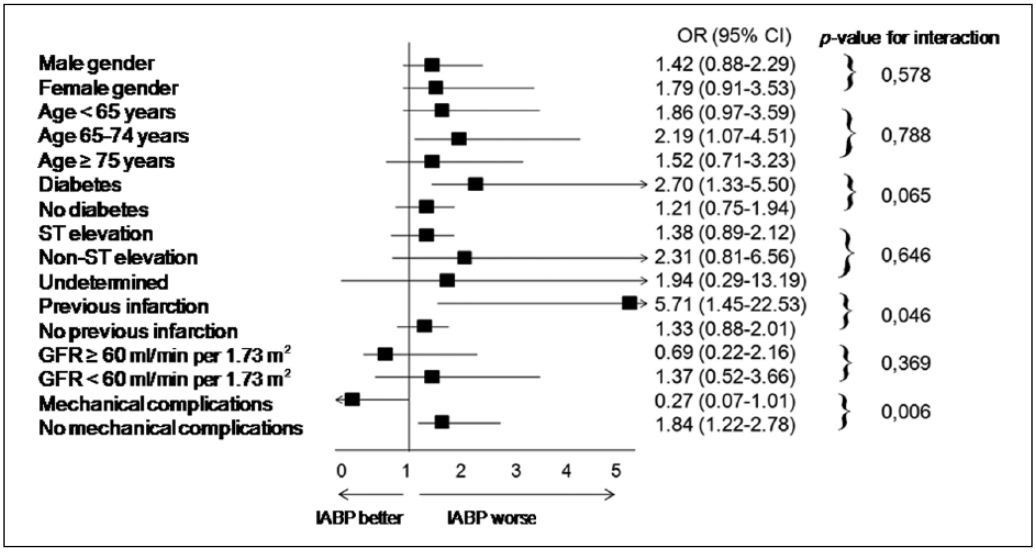

Figure 3. Subgroup analysis for the composite end point (in-hospital all-cause mortality and re-infarction).
OR: odds ratio; CI: confidence interval; GFR: glomerular filtration rate; IABP: intra-aortic balloon pump.

p=0.057). The composite end point of all-cause death/re-infarction was 42.4% in the overall population, significantly higher in the IABP group (OR 1.53, 95% CI 1.03–2.25, p=0.032). All other secondary end points were similar between groups. These results (all-cause mortality and the composite end point) were consistent in all specified subgroups (Figures 2 and 3) with the exception of patients with previous MI, who had worst outcome with IABP, and patients with mechanical complications, with better outcome with IABP use.

Table 3 details the multivariate analysis with the independent predictors of all-cause in-hospital mortality and the composite end point death/re-infarction. IABP use was independently associated with worst in-hospital outcome. Age and previous stroke/TIA were also independent predictors of outcome. Smoking had a paradoxical beneficial effect in outcome.

Survival analysis at six months showed that the use of IABP was not an independent predictor of all-cause mortality (HR 1.138, 95% CI 0.855–1.515), even after adjustment in a multivariate model (HR 1.141, 95% CI 0.836–1.559) (Figure 4(a)).

Safety

Safety end points results are shown in Table 2. In the entire study group, stroke/TIA rate was 2.2% and major bleeding occurred in 6.7% of the patients. No significant differences

were detected between the IABP group and the group that did not receive IABP with respect to the rates of stroke/TIA or major bleeding.

Propensity score-matched models

After propensity score matching, patients' characteristics were similar between both groups (Table 4). In this matched cohort, death occurred in 39.8% of patients in the group without IABP and 40.8% in the group with IABP (OR 1.04, 95% CI 0.63–1.74, p=0.865). For the composite outcome of death/re-infarction, it occurred in 41.0% *vs*. 46.9% respectively (OR 1.27, 95% CI 0.77–2.11, p=0.349).

Table 3. Multivariate logistic regression analysis for in-hospital all-cause mortality and in hospital death/re-infarction.

	OR	95% CI	*p*-value
	In-hospital all-cause death		
Age, per 10 years	1.52	1.28–1.80	<0.001
Smoking	0.59	0.36–0.95	0.031
Previous stroke/TIA	2.01	1.11–3.64	0.021
IABP	1.77	1.15–2.73	0.010
	In-hospital death/re-infarction		
Age, per 10 years	1.49	1.26–1.76	0.040
Smoking	0.62	0.39–0.99	0.046
Previous stroke/TIA	1.81	1.00–3.28	0.049
IABP	2.01	1.31–3.09	0.001

OR: odds ratio; CI; confidence interval; IABP: intra-aortic balloon pump; TIA: transient ischaemic attack.

In survival analysis, the outcome was identical between patients with and without IABP (Figure 4(b)). Also in a Cox proportional hazards model, IABP was not associated with a different outcome (adjusted HR 0.996, 95% CI 0.674–1.471).

Discussion

In our large nationwide registry of patients with acute MI, the use of IABP was not associated with a reduction in in-hospital all-cause mortality/re-infarction or six-month all-cause mortality in the subgroup of patients with cardiogenic shock complicating MI for whom early revascularization was planned. These results are also consistent in a subgroup analysis, with the exception of patients with mechanical complications, where IABP was associated with improved in-hospital outcome.

Our results were further consolidated by the use of propensity score matching, which allowed an adequate balance of baseline and treatment characteristics. The absence of benefit with IABP after propensity score matching analysis remained consistent with our short-term estimates.

In previous registries, which represent real-world treatment, the rate of cardiogenic shock in populations with ACS ranged from 1% to 4.6%.[1,2] Our registry showed a rate of 4.2%, similar to other registries. Although rare, cardiogenic shock is usually a deadly complication of acute myocardial infarction. The mortality rate of 40.2% in our study population was similar to recent trials, and slightly better than the 42% to 48% mortality rate reported in older randomized trials and registries.[1,2,7] Other authors explained this lower rate in recent studies with the possible inclusion of a higher percentage of patients with mild or moderately severe cardiogenic

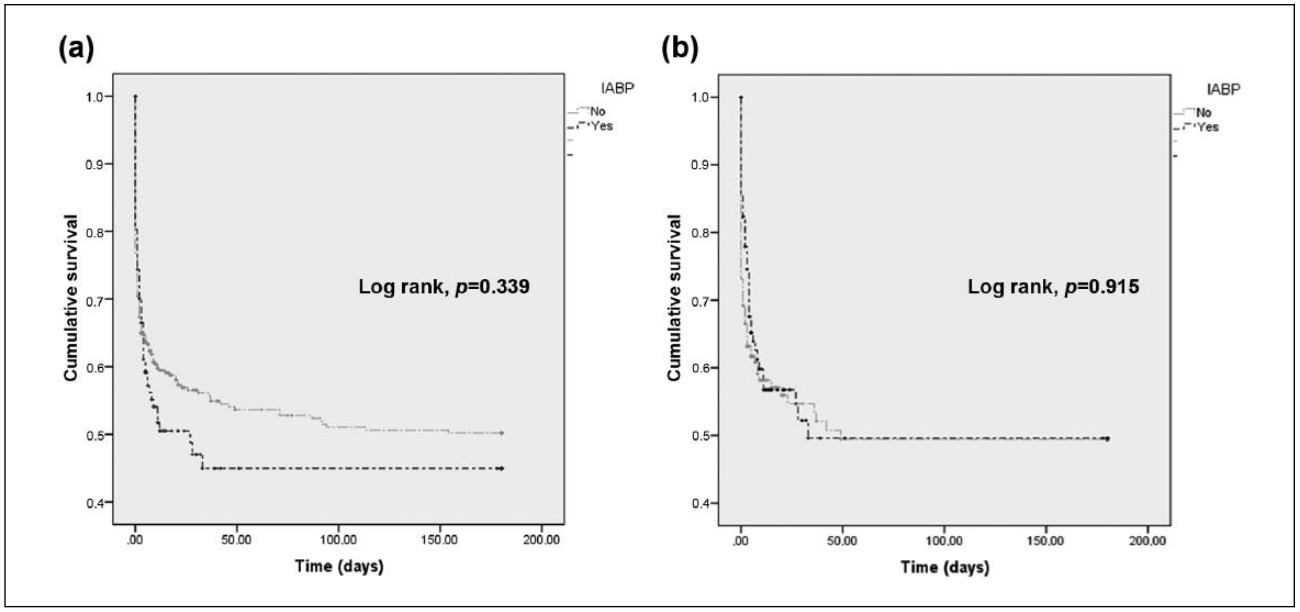

Figure 4. Kaplan–Meier survival analysis comparing patients with and without intra-aortic balloon pump (IABP). (a) Overall study population; (b) propensity score matched population.

Table 4. Clinical characteristics by study group after propensity score matching.

	Without IABP *n*=161	With IABP *n*=98	*p*-value
Age, years	66 ± 13	66 ± 12	0.979
Male gender, %	65.8	65.3	0.930
BMI, kg/m^2	27.1 (24.7–29.7)	26.2 (24.5–28.3)	0.073
Risk factors, %			
Hypertension	64.0	64.3	0.960
Hyperlipidaemia	49.7	51.0	0.835
Diabetes	33.5	34.7	0.849
Smoking	29.2	28.2	0.644
Previous history, %			
Myocardial infarction	8.7	9.2	0.893
PCI	6.8	8.2	0.690
CABG	0	1.0	0.378
Stroke/TIA	9.3	13.3	0.321
PAD	3.1	3.1	1.000
Initial presentation			
EMS, %	10.6	11.2	0.867
Heart rate, beats/min	88 ± 28	88 ± 29	0.888
SBP, mmHg	105 ± 31	104 ± 31	0.791
Killip class IV, admission, %	42.2	47.4	0.424
STEMI, %	82.0	76.5	0.414
Anterior	67.4	69.3	0.777
Thrombolysis	14.5	9.1	0.287
GRACE risk score	175 (158–203)	193 (154–218)	0.418
Multivessel disease, %	65.2	63.3	0.750
Left main disease, %	19.3	24.5	0.318
Treatment, %			
ASA	96.3	98.0	0.714
Clopidogrel	81.4	86.7	0.260
ACEI	47.6	55.1	0.218
Beta-blocker	39.1	43.9	0.415
Statin	72.0	76.5	0.427
PCI	86.3	86.7	0.927
CABG	1.2	3.1	0.107
Mechanical complications, %	11.2	11.2	0.991
Stroke /TIA, %	2.5	3.1	1.000
Major bleeding, %	5.6	8.2	0.417
In-hospital death, %	39.8	40.8	0.865
In-hospital re-infarction, %	3.1	8.2	0.083
Death/re-infarction, %	41.0	46.9	0.349

IABP: intra-aortic balloon pump; BMI: body mass index; PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting; TIA: transient ischaemic attack; PAD: peripheral arterial disease; SBP: systolic blood pressure; EMS: emergency medical system; STEMI: ST elevation myocardial infarction; GRACE: Global Registry of Acute Coronary Events; ASA: acetyl salicylic acid; ACEI: angiotensin converting enzyme inhibitor.

shock. However, our results being similar seems to support the fact that they were obtained in a population of patients with contemporary treatment and better outcomes. In fact, only patients with a planned revascularization were included in our study as well as in the IABP-SHOCK 2 trial. If we consider all the patients in our registry with cardiogenic shock, including patients that were not submitted to early invasive strategy, hospital mortality was 49.8%, confirming the benefit of early revascularization in these patients.

Previous studies showed that IABP is a potentially valuable therapy for circulatory support in cardiogenic shock because it ameliorates ischaemia by simultaneously augmenting coronary blood flow and reducing myocardial oxygen demand.[12,13] However, despite the limited evidence by randomized controlled trials, IABP is the most commonly used mechanical assistance device for patients in cardiogenic shock due to acute myocardial infarction. Until recently, guidelines from the American Heart Association/American College of Cardiology and from the European Society of Cardiology strongly recommended the use of IABP as a class I recommendation for the treatment of patients with infarct-related cardiogenic shock.[3,4] However, that strategy was developed in the era of thrombolytic treatment of acute myocardial infarction. At that time, the SHOCK trial showed that thrombolytic therapy, IABP and revascularization by PCI/CABG were associated with

lower in-hospital mortality rates. A strategy of early thrombolysis and IABP was particularly recommended in hospitals without revascularization facilities, followed by immediate transfer for coronary angiography.[14] This benefit was subsequently supported by a 12-month analysis that favoured IABP after thrombolytic therapy in cardiogenic shock complicating myocardial infarction, particularly when early revascularization was not available.[15] Also patients that underwent emergent revascularization but did not receive previous thrombolytic therapy had lower 12-month mortality with IABP (42% *vs*. 60%).

The GRACE registry showed that 4.6% of patients with ACS developed cardiogenic shock.[1,2] From these, 57% underwent coronary angiography and only 47% coronary revascularization. In our registry, only 43.6% underwent early coronary angiography. However, the revascularization rate in the group submitted to coronary angiography was very high (83.4% had successful PCI and 2.4% CABG). In the Euro Heart Survey, only 25% of acute MIs complicated by cardiogenic shock undergoing PCI were treated with IABP.[16] In our registry, that rate was even lower.

In a more contemporary treatment era, where emergent revascularization is the cornerstone treatment to improve survival, evidence does not seem to favour IABP use. Both the Euro Heart Survey on Percutaneous Coronary Intervention data analysis and several meta-analyses showed that the use of IABP was not associated with any survival improvement.[5,16] This subject was addressed in the IABP-SHOCK 2 trial, which showed no benefit with the use of IABP in patients with cardiogenic shock in the contemporary era of MI treatment.[7] There was no immediate improvement in haemodynamic status or in the systemic inflammatory response syndrome. Although there was a positive effect of IABP on multiorgan dysfunction in the first few days, this effect was no longer present at day four. These results were further supported at 12-month follow-up, with no benefit of IABP in all-cause mortality (HR 1.01 95% CI 0.86–1.18, p=0.91) or re-infarction (HR 2.60, 95% CI 0.95–7.10, p=0.05).[17] However, these results do not reflect real-world treatment and the translation to real-life populations is not always adequate due to different characteristics of patient populations and centres.

We decided to study the role of IABP treatment in a large national registry of ACS in patients with acute MI complicated by cardiogenic shock. This registry expresses contemporary treatment in real life, and we sought to study the potential beneficial or harmful effects of IABP. We showed that IABP use was not associated with any improvement in outcome in patients with MI-related cardiogenic shock. Only in patients with mechanical complications did IABP improve survival and reduce the composite end point of death/re-infarction. Thus, our study is in line with previous trial, registries and meta-analyses[5–7,18–21] and supports the recent downgrading of IABP indication in patients with MI-related cardiogenic shock.[18,19] In these guidelines, IABP is a class IIIA recommendation for routine use in acute MI complicated by cardiogenic shock and a class IIaC recommendation in the case of cardiogenic shock due to mechanical complications.

Particularly interesting was the finding that patients with previous MI had a more unfavourable outcome with IABP support compared with those without previous MI. There was no evident explanation, except from the longer symptom to admission time (134 min *vs*. 68 min, p=0.09), lower frequency of prior cardiac medication use and previous revascularization, and higher frequency of STEMI and left main stenosis (Supplementary Material Table 2 online).

The timing of IABP deployment could be important but we did not address that subject in our study. However, a recent analysis of the Swedish Coronary Angiography and Angioplasty Registry (SCCAR) showed that the timing of IABP insertion (before or after primary PCI) was not associated with a different outcome.[22]

Limitations

Our study is an observational and non-randomized study. Although most Portuguese centres participated in the registry, at different treatment levels, not all were included.

Only patients with quick access to catheterization facilities and who were submitted to urgent coronary angiography, with IABP availability, were included in the analysis. Coronary revascularization has important implications in outcome and we tried to obtain a homogeneous population with very specific inclusion criteria.

In our registry, we lack complete information on the type and timing of mechanical complications and subsequent treatment. Patients with cardiogenic shock and mechanical complications had a significantly higher hospital mortality (55.0% *vs*. 40.2%, p=0.038). We also found that only 20% of the patients with MI and a mechanical complication were treated with IABP, with a mortality rate of 25.0% compared with 62.5% in the group without IABP (p=0.020). A detailed analysis on the type of mechanical complication and specific treatment would be important to explain the rate of IABP use and the benefits associated with its use. There might have been some treatment bias from the attending physician when choosing to use IABP. This is an interesting subject to study in the future, to understand whether IABP is more used in patients with specific types of mechanical complications or in patients submitted to medical or surgical treatment. Also in our study, we only considered patients with early cardiogenic shock and early invasive strategy including IABP in the first 24 h. However, some mechanical complications are usually late complications and later IABP use was not captured in our study. Despite this, we believe that most complications occurred early and were the cause of cardiogenic shock.

Unexpectedly, we found a similar rate of stroke/TIA and bleeding in both study groups. A possible explanation was that our registry could not be capable of capturing those adverse events. However, the definitions were very objective and it does not seem to be the case. If we had considered also

minor bleedings, the results would probably have been worse for IABP, but we decided not to use that end point because minor bleeding definition is more subjective and it might be incorrectly represented in the registry.

Conclusions

In a large population of patients with acute MI complicated by cardiogenic shock, IABP was used in less than one-fifth of patients submitted to coronary angiography. In this nationwide registry, IABP use was associated with a neutral effect in outcome. Only patients with mechanical complications had significant benefits in survival with IABP use.

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Conflict of interest

None declared.

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